ALPHA-1-ANTITRYPSIN LEVEL IN EGYPTIAN ASTHMATIC CHILDREN

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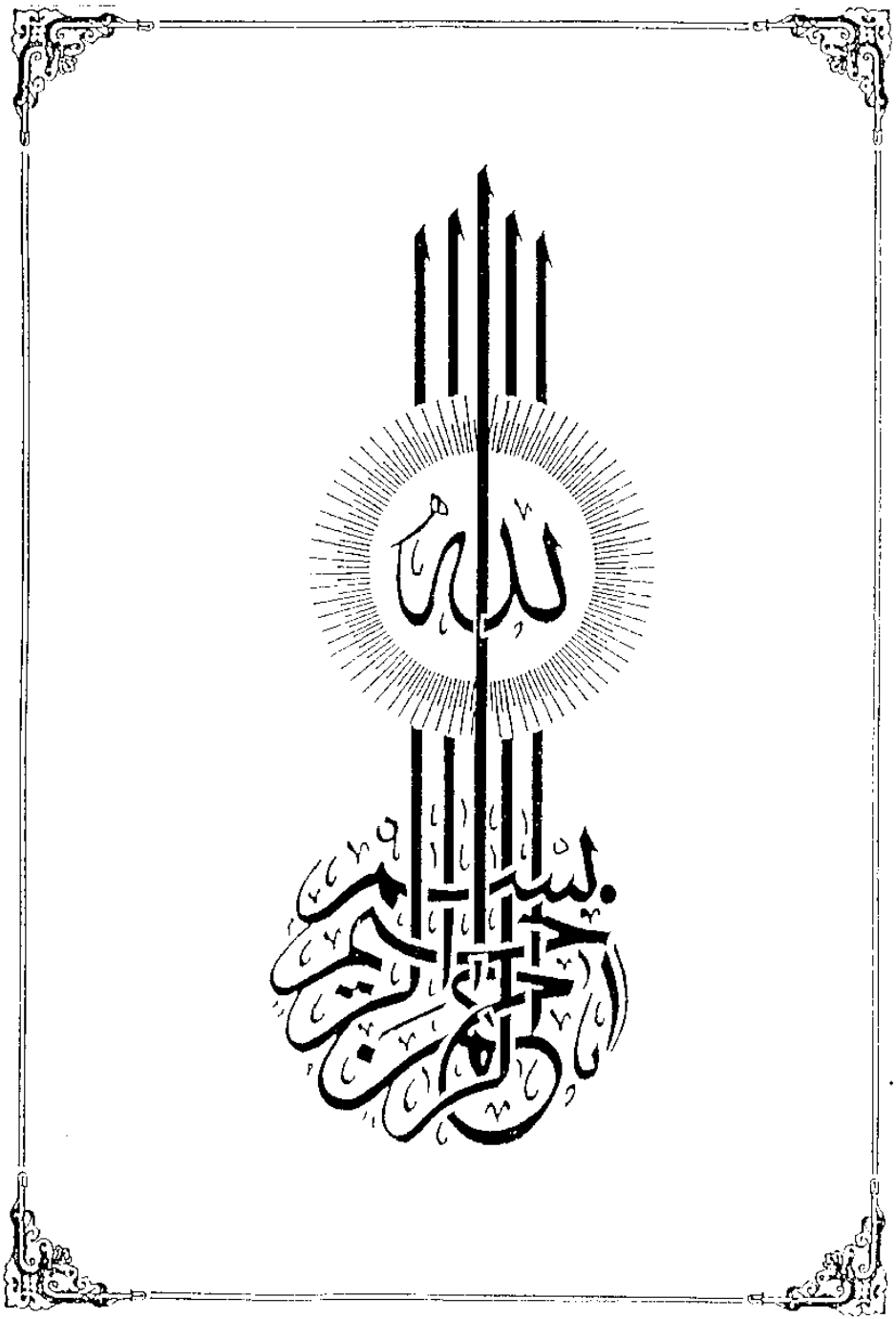

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ABBREVIATIONS

$\&_1$-AT = Alpha-1-Antitrypsin.

$\&_1$-PI = Alpha-1-Protease inhibitor.

Cylic-GMP= Cylic - Guanosine Mono Phosphate.

GOAD = Generalized Obstructive Airway Diseaes.

IDL = Intermediate density lipoproteins.

LDL = Low density lipoporteins.

Pi = Protease inhibitor.

STIC = Serum Trypsin Inhibitory Capacity.

VLDL = Very low density lipoproteins.

Cyclic-AMP Cyclic Adenosine Mono Phosphate.

Introduction
&
AIM OF WORK

INTRODUCTION

The word Asthma (Greek - breathe hard) has retained its meaning intact through the centuries, although the pathogenesis of the disease remains poorly understood.

Many trigger mechanisms provoking episodes of asthma are well understood, but there are others that are not known (Kuzemko, 1976).

Asthma is a common problem in childhood. In a study of hospital admissions in Pittsburgh, U.S.A. Fireman (1971) showed that the number of children with asthma admitted to hospital was doubled in recent years and the severity of the disease had increased considerably during the last ten years. In general, up to the age of 15 years about 2-3% of boys and 1-2% of girls have asthma (Rhyne, 1974).

Alpha-1-antitrypsin is a glycoprotein with a molecular weight of about 52.000, it consists of a single polypeptide chain with a number of carbohydrate side chains.

Studies with acid-starch gel electrophoresis showed that alpha-1-antitrypsin exists in a number of biochemical forms or variants, which are known collectively as the Pi system, the abbreviation standing for protease inhibitor.

Each variant is labelled according to its mobility in an acid-starch gel as follows:-
S = indicates slow, Z = very slow, M = medium, and F = fast (Tobin et al., 1982).

The capacity of human serum to inhibit the proteolytic activity of trypsin was recognized by Camus and Gley in 1897, but, it was not until 1963, when Laurell and Eriksson, reported the homozygous deficiency of alpha-1-antitrypsin. Eriksson later in 1965, discovered the association between homozygous deficiency of alpha-1-antitrypsin and the development of chronic obstructive pulmonary disease.

Alpha-1-antitrypsin deficiency is a disease inherited in a multiallelic, autosomal, codominant pattern (Fagerhol, and Laurell, 1967).

Recent studies have raised the question whether obstructive pulmonary diseases other than emphysema and bronchitis may also be linked to a deficiency in alpha-1-antitrypsin.

Fagerhol and Hauge (1969); Hyde et al (1972); and others made such studies with variable results.

AIM OF THE WORK

The aim of our work is to assess the level of alpha-1-antitrypsin enzyme in the serum of Egyptian Asthmatic Children.

The level of cholesterol and triglycerides in the serum of the same patients and control were also assessed to study the affection of fat metabolism in bronchial asthma.

Review of Literature

REVIEW OF LITERATURE

BRONCHIAL ASTHMA

Definition:

In children, asthma is defined in clinical and functional terms as a condition of altered dynamic state of respiratory passages due to the action of diverse stimuli resulting in airways obstruction of varying degree and duration, and reversible partially or completely spontaneously or under treatment (Kuzemko, 1976).

Incidence and Prevalence:

The true incidence of asthma in children remains unknown, in general, up to the age of 15 years about 2-3% of boys and 1-2% of girls have asthma (Rhyne, 1974).

Dodge and Burrows (1980) in their study reported that the incidence of asthma was greatest in young children, least in late adolesence and increases again in early adult life. The incidence was much greater in women older than 40 years of age and it was 1.5 times more in young boys than in young girls.

ETIOLOGY OF BRONCHIAL ASTHMA

The asthmatic attack is caused by obstruction of the airways by spasm, oedema and inflammation.

Any factor which can cause imbalance in the neural and humoral forces may cause bronchial asthma. Bronchial asthma is a complex disorder involving biochemical, autonomic, immunologic, infectious, endocrinal and psychological factors in varying degrees in different individuals (Vaughan et al., 1981).

The factors which determine the incidence and prevalence of asthma and appear to precipitate the attacks are either related to the patient or to the environment.

A- Factors Related to the Patient:-

1- Age of onset:

More than 75% of asthmatic children have their first symptoms before the age of five years (Dawson et al., 1969).

2- **Sex:**

Before puberty asthma is more prevalent in males than in females, but after pubety sex incidence is equal.

Atopic asthma is more prevalent among men than among women, and non-atopic asthma occurs more often in women than in men (williams et al., 1969).

3- **Genetic factors and Familial tendency:**

A family history of asthma or other manifestations of hypersensitivity states are often present in about 50 to 75 per cent of cases.

The data up to that time agree with a polygenic or multifactorial inheritence for asthma, but non-genetic factors are also important in the production of asthma (Cohen 1974).

4- **Past history:**

Many children with eczema developed bronchial asthma (Zach et al, 1981). A past history of bronchiolitis is also an important factor in developing bronchial asthma (Horn et al, 1975).